APPENDIX A

The numbers assigned to the following article summaries correspond with the numbers assigned to the synopses of the articles in the text of this Report.

RESPIRATORY DISEASES AND CONDITIONS – CHILDREN

[28] **"Passive Smoking as a Determinant of Bronchial Responsiveness in Children," F. Forastiere, N. Agabiti, G.M. Corbo, R. Pistelli, V. Dell'Orco, G. Ciappi and C.A. Perucci, *American Journal of Respiratory and Critical Care Medicine* 149: 365-370, 1994**

"The present study was...designed to clarify the effects of parental smoking on the degree of nonspecific bronchial responsiveness in children; factors that may reflect the child's level of exposure to environmental tobacco smoke were also considered."

"There were 825 children exposed to any parental smoke; in this group, in 14.4% only the mother smoked, in 49.0% only the father smoked, and in 36.6% both were smokers...A low level of father's education was more prevalent among the group of subjects exposed to parental smoking, but no other differences were noteworthy."

"As in the larger data set, in the subsample maternal smoking was related to increased risk of asthma (OR 1.48, 95% CI 0.84 to 2.65) and cough or phlegm (OR 1.75, 95% CI 0.87 to 3.56), even though in this case the associations were not statistically significant. Sex-, height-, and age-adjusted FEV_1 and FEF_{25-75} were lower among subjects with passive smoking exposure in comparison with nonexposed individuals."

"In boys, there was no statistically significant increased risk in the categories studied, even though nonsignificant elevated odds ratios were found for both maternal and paternal smoking among those living in high-density households."

"For girls, both maternal and paternal smoking had a deleterious effect; statistically significant higher odds ratios were found for all responders (any smokers, OR 1.50; maternal smoking, OR 1.58) and for strong responders (any smokers, OR 2.70; maternal smoking, OR 2.92; paternal smoking, OR 2.59)."

"Gender, however, appears to modify the effect of passive smoking on BR in our study. Females had a greater frequency of response to methacholine than males when exposed to parental smoking."

"The simplest explanation for our results in girls is that they spend more time at home and are more exposed. An alternative possibility is that girls are more susceptible."

"The mechanism by which passive smoking can affect BR is unclear. Side-stream tobacco smoke, because of its complex chemical nature, may elicit an irritant effect involving parasympathetic receptors and a local inflammatory response of the bronchial tree, thus directly increasing BR; however, other pathways have been hypothesized."

"In conclusion, we suggest that the effects of parental smoking on children's BR are detectable when the conditions for a higher exposure level at home are met. Females seem to be more susceptible. The findings reinforce the evidence of an association between passive smoking and inception of bronchial asthma."

OTHER HEALTH ISSUES

[29] **"Passive Smoking During Pregnancy and the Risk of Delivering a Small-for-Gestational-Age Infant," I. Fortier, S. Marcoux, and J. Brisson, *American Journal of Epidemiology* 139(3): 294-301, 1994**

"The objective of our analysis was to further assess the relation between maternal passive smoking during pregnancy and the risk of delivering a small-for-gestational-age (SGA) infant. We attempted to overcome some of the limitations of the previous studies in the following ways: our exposure data pertain to passive smoking at home and in the workplace, the associations are described for each surrounding separately, the analysis controls for all known confounders, including selected job characteristics, and the dose-response relation between passive smoking and the risk of SGA is also assessed."

"Among nonsmokers, 49 percent were exposed to environmental tobacco smoke, either at home only (13

percent), at work only (28 percent), or in both surroundings (8 percent)....Passive smokers included a larger proportion of nulliparae and tended to have higher caffeine intakes than other nonsmokers. Among women who had a paid occupation, passive smokers were more likely to work on evenings or nights and to have jobs involving lifting or long standing periods than unexposed women."

"Overall, nonsmokers passively exposed to tobacco smoke were at little or no higher risk of delivering a SGA infant (OR = 1.09, 95 percent confidence interval (CI) 0.85-1.39) than unexposed women. Passive smoking at home only was not related to SGA (OR = 0.98). . . . [B]eing passively exposed at work only yielded an adjusted odds ratio of 1.18 (95 percent CI 0.90-1.56). The relation between SGA and passive smoking at work only was further examined. The risks of SGA increased slightly but consistently when the weekly hours of exposure, the number of weeks of exposure during pregnancy, and the subjective intensity of exposure increased. However, none of the trends was statistically significant."

"We observed a modest increase in the risk of delivering a SGA infant in pregnant nonsmoking mothers exposed to passive smoking in the workplace. The point estimates increased consistently with the duration and the intensity of exposure, which supports the hypothesis that passive smoking during pregnancy may affect fetal growth. However, our results need to be interpreted with caution because the associations are weak, the confidence intervals all include null values, the tests for trends are not conclusive, and the association is not found in women exposed to smokers at home only."

"[T]he exposure may indeed be more important in the workplace than at home since the number of smokers and the environmental conditions (e.g., ventilation, room size, insulation) may differ in these two surroundings...[A]s the measure used to assess exposure at home (smoking habit of family members) differs from that used to assess exposure at work (hours of exposure), results may not be comparable. . . . [S]moking habit of family members is thought to be a less accurate measure than is the duration of exposure. If so, the associations with passive smoking at home would be more likely to be underestimated due to misclassification."

"Given the difficulty of documenting small effects in epidemiologic studies, in our view, future investigations are not likely to clarify the relation of passive smoking to SGA unless they are restricted to nonsmokers, are based on large prospective cohorts, document exposure in the several possible environments and in different periods of pregnancy, and include a biologic marker of exposure as a supplement to detailed questionnaire data."

[30] "Environmental Tobacco Smoke: The Risks of Passive Smoking in Facial Surgery," A. Matarasso, *Annals of Plastic Surgery* 31: 573, 1993

"In plastic surgery, the harmful effects of *primary* cigarette smoking...following surgery and during wound healing have been well documented...What has not been subject to review are the effects of *passive smoking.* One could extrapolate from what is already known about passive smoking and its effects on patients to speculate on instances of exposure to environmental tobacco smoke (ETS) and to take similar, necessary precautions."

"A couple that had been together for 44 years — living and working — recently underwent facial rejuvenation surgery on the same day. The husband's cigarette use consisted of more than 100 packs per year....In the early postoperative period, [the nonsmoking wife] experienced areas of skin ischemia, resulting in superficial epidermolysis and pigmentary changes."

"Environmental tobacco smoke results in involuntary exposure derived from 'mainstream' and 'sidestream' smoke...Although the observations here are anecdotal and wound healing is a multifactorial process, in view of what has been clearly established regarding the adverse effects of primary cigarette smoking, the preponderance of evidence suggests that it is advisable to consider ETS as a possible deleterious factor. When screening patients and operating, the effect of ETS on delayed wound healing or tissue necrosis may in fact be an additional risk factor that can confound procedures that necessitate mobilization of flaps, and should not be underestimated."

[31] **"Intellectual Impairment in Children of Women Who Smoke Cigarettes During Pregnancy," D.L. Olds, C.R. Henderson, and R. Tatelbaum, *Pediatrics* 93: 221-227, 1994**

"In the current paper we examine whether the relationship between maternal prenatal cigarette smoking and children's intellectual impairment during the first 4 years of life remains after controlling for a wide range of potentially confounding influences."

"Interviews with the mother were conducted at registration during pregnancy, at the 34th week of gestation, and at 6, 10, 22, 34, and 46 months of the child's life."

"When smoking was defined by level of postnatal (46-month) smoking, the adjusted difference in children's IQs (averaged across 3 and 4 years of age) between those whose mothers smoked 0 and those whose mothers smoked 10+ cigarettes per day was 3.09 points (95% CI: - 0.93, 7.11)...[T]he greatest difference in children's intellectual functioning was found for cigarette smoking measured at the end of pregnancy."

"Children born to women who smoked heavily during pregnancy (10+ cigarettes per day), and who did not receive nurse-visitation services, had IQ scores at 1 and 2 years of age that were nearly 7 points lower, and at 36 and 48 months that were 9 points lower than children born to women who did not smoke during pregnancy. These differences were explained in part by associated differences in social class, maternal education, IQ, qualities of caregiving, and conditions in the home environment. Even after control for these biasing influences, however, a significant difference of 4.35 IQ points remained at 3 and 4 years between the children of women who smoked substantially versus those who did not smoke at all during pregnancy. This four-point effect, although small, is comparable with the adverse influence of low levels of lead exposure on preschoolers' IQ test performance where the children do not show symptoms of lead encephalopathy."

"[W]e did not assess fully the child's exposure to side-stream smoke during the first 4 years after delivery. It is revealing, nevertheless, that maternal post partum smoking was less predictive of the children's intellectual development than was prenatal smoking. We assume that a substantial portion of the children's exposure to passive smoke is likely to come from the mother, and that where the mother smokes other individuals in the household also are likely to smoke. Clearly, more work needs to be performed on this topic, using more valid and reliable measures of passive smoking."

"Moreover, it is clear that women who smoked 10+ cigarettes per day were substantially different from those who did not smoke at all. It is possible that we did not measure all possible confounders and that some other unidentified variable accounts for the differences in children's IQs associated with maternal prenatal smoking levels."

ETS Exposure and Monitoring

[32] **"Passive Exposure to Tobacco Smoke in Children Aged 5-7 Years: Individual, Family, and Community Factors," D.G. Cook, P.H. Whincup, M.J. Jarvis, D.P. Strachan, O. Papacosta, and A. Bryant, *British Medical Journal* 308: 384-389, 1994**

"We present an analysis of the relation between cotinine concentration and questionnaire data in children aged 5-7 from 10 towns in England and Wales. We examine the importance of parental exposure as opposed to other sources; exposure among children from non-smoking households; and the social and geographical pattern of passive exposure to tobacco smoke in children."

"The study was carried out in 10 towns in England and Wales — five with high adult cardiovascular mortality and five with low mortality."

"Fifty three percent (1610/4030) of children were exposed to at least one smoker."

"Geometric mean cotinine concentration varied greatly with source of exposure from 0.29 ng/ml in children with no identified source of exposure to 4.05 ng/ml when both parents smoked, a 13.7 fold increase. In the 20 children in whom both parents smoked more than 20 cigarettes a day the geometric mean cotinine was 9.03 ng/ml (95% confidence interval 6.73 to 12.1)."

"Our data confirm that parental smoking is the most important source of passive exposure to smoke in young children and show a clear dose response with number of cigarettes smoked a day. While mothers were less likely to smoke than fathers, the effect on cotinine concentrations when they did so was greater, presumably because they spend more time with the children. The difference in effect was small at low levels of smoking, but pronounced at higher levels. One interpretation is that fathers who smoke heavily are less likely to do so in the presence of the child than mothers who smoke heavily. Overall, maternal smoking contributed more to the children's burden of cotinine than did paternal smoking. Other people smoking in the household and being looked after by someone from outside the household who smoked also made small contributions to exposure. However, such sources of exposure were relatively uncommon and when present were less important than parental smoking."

"[N]icotine is not entirely specific to tobacco. It is also found in small amounts in peppers, aubergines, and potato skins. We have previously argued that these are unlikely to greatly influence cotinine concentration. The uniformly low concentrations among our non-exposed children suggest that either the higher concentrations seen in the exposed children are due to smoking or the dietary factors are almost entirely confounded with smoke exposure in the home, which seems implausible. Even among our non-exposed children the concentrations of cotinine correlate with community smoking habits, which suggests that any other sources of cotinine make only a very small contribution."

"The social class and geographical differences in cotinine concentrations emphasize the variation in passive exposure to tobacco smoke among children from different backgrounds."

"Though the identified sources of exposure were the most important determinants of variation in cotinine concentrations, other sources and modifying factors clearly existed. Eighty eight percent of children not exposed at home and not looked after by a smoker had cotinine detected in their saliva. Cotinine concentration in non-exposed children was related to both social class and town of residence and was presumably attributable to sources we did not inquire about. This is supported by the finding that the cotinine concentrations in non-exposed children were directly related to the community level of smoking."

"7-11% of the population burden of cotinine was in children not exposed to any of the sources we asked about. The correlation between cotinine concentrations in such children and the prevalence of smoking in the community suggests that passive smoking should be viewed as a community exposure rather than simply as an aspect of family lifestyle."

[33] "Hair Concentrations of Nicotine and Cotinine in Women and Their Newborn Infants," C. Eliopoulos, J. Klein, M.K. Phan, B. Knie, M. Greenwald, D. Chitayat, and G. Koren, *Journal of the American Medical Association* 271(8): 621-623, 1994

"In the present study we describe the distribution characteristics of nicotine and its major metabolite, cotinine, in maternal and neonatal hair. These data suggest that accumulation of nicotine and cotinine in neonatal hair may be used clinically and in laboratory studies to estimate fetal exposure to maternal cigarette smoking."

"Mothers who were active smokers, passive smokers, or nonsmokers were identified in two newborn nurseries in Toronto, Ontario, 1 to 3 days after delivery. . . . Passive smoking was defined as regular and steady gestational exposure to other persons' cigarette smoke, either at home (eg, smoking by husband or partner) or in the workplace. Hair samples were obtained by cutting five to seven hair shafts."

"Thirty-five nonsmoking mothers participated in the study. Their mean hair concentrations of nicotine and cotinine were significantly lower than in smoking mothers. Similarly, neonatal hair concentrations of nicotine and cotinine were significantly lower in infants of nonsmokers than in infants of smokers."

"Twenty-three passive smoking mothers and their infants participated in the study. Their concentrations of nicotine and cotinine were intermediate and significantly different from those of both the smokers and nonsmokers."

"[O]ur results suggest that maternal and fetal hair may better estimate long-term systemic exposure to the

toxic constituents of cigarettes than the reported consumed dose and thus may yield a better prediction of fetal/neonatal risk."

"During the last few years there has been increasing awareness of the serious health risks inflicted by passive exposure to cigarette smoke. In a recent study, Makin et al. documented impaired neurodevelopment in infants exposed in utero to passive maternal smoking compared with nonsmoking controls. Analysis of such data is complicated because of the potential confounding variables in assessing toddler achievements in cognitive tests; however, these researchers controlled for known confounders in their model. On the other hand, the degree of fetal exposure to cigarette smoke could not be evaluated in the absence of a biological marker. Our data indicate that, indeed, passive smoking pregnant women and their infants accumulate nicotine and cotinine to measurable levels. These data confirm our preliminary findings in four passive smokers who were initially included in a group of nonsmokers. Because hair accumulation of cotinine reflects long-term exposure, it may provide a more accurate determination of fetal exposure to cigarette smoke than the reported number of cigarettes consumed."

[34] **"Reduced Plasma Ascorbic Acid Concentrations in Nonsmokers Regularly Exposed to Environmental Tobacco Smoke," D.L. Tribble, L.J. Giuliano, and S.P. Fortmann, *American Journal of Clinical Nutrition* 58: 886-890, 1993**

"Radical-mediated oxidative processes have been implicated in the pathogenesis of chronic and degenerative diseases including cancer and atherosclerotic heart disease....Smokers exhibit reduced concentrations of the antioxidant vitamin ascorbic acid (AA)."

"Environmental tobacco smoke (ETS) is now recognized to predispose exposed individuals to the development of chronic diseases, although little is known about the nature and magnitude of the pathophysiological effects of ETS exposure, or the degree to which these may contribute to increased disease risk. Sidestream smoke contains a greater oxidant load than mainstream smoke and high ambient concentrations of oxidants are likely to be present in inadequately ventilated homes, workplaces, and other enclosed settings where smoking is permitted. We hypothesized that nonsmokers regularly exposed to such conditions were chronically oxidatively stressed and thus would exhibit aberrations in AA nutriture similar to smokers. To examine this possibility we compared plasma AA concentrations and vitamin C intakes in a cross-sectional sample of women classified as either active heavy smokers (AS), regular passive smokers (PS), or nonexposed nonsmokers (NNS)."

"Our results confirm previous reports of reduced plasma AA concentrations in AS and additionally show that PS exhibit reduced concentrations relative to NNS."

"In contrast to observations of decreased vitamin C consumption in smokers, we did not observe decreased vitamin C intakes in smoke-exposed populations relative to NNS."

"Reduced plasma AA concentrations were apparent even in AS reporting dietary vitamin C intakes exceeding current recommended dietary allowances (RDAs) for smokers. These data support previous contentions that dispute the recent increase to 100 mg vitamin C/d, the RDA is still inadequate for smokers...Our results additionally suggest that current vitamin C intake recommendations are inadequate to meet the increased needs of nonsmokers regularly exposed to ETS. Plasma AA concentrations were reduced in PS reporting vitamin C intakes up to 250 mg/d, but not at higher intakes."

"In summary, we observed reduced plasma AA concentrations in AS and PS relative to NNS that were not attributable to differences in vitamin C intakes among these populations. These results suggest that, like AS, PS are subjected to severe oxidant exposure. Oxidative mechanisms have been implicated in numerous chronic and degenerative diseases and thus may be involved in the increased risk associated with ETS exposure. Although reductions in plasma AA concentrations in PS may be overcome at higher vitamin C intakes, this is unlikely to completely ameliorate the pathogenic consequences of the underlying oxidant pressure."

STATISTICS AND RISK ASSESSMENT

[35] **"Sponsored Symposia on Environmental Tobacco Smoke," L.A. Bero, A. Galbraith, and D. Rennie, *Journal of the American Medical Association* 271(8): 612-617, 1994**

"To suggest substantial support for their position that ETS is not harmful, the tobacco industry frequently cites industry-funded, non-peer-reviewed publications, such as symposium proceedings. This study examines symposia on ETS to determine whether they should be considered equivalent to other sources of scientific information on ETS. This study was designed to examine the extent of tobacco industry sponsorship of symposia on ETS; to see whether there was a balanced presentation of the data on ETS in such symposia compared with articles on ETS in the peer-reviewed literature; to test whether these symposia represented the work of tobacco industry-sponsored scientists relatively more than did articles on ETS in the peer-reviewed journals; and to compare the symposium authors and their sponsorship with the authors of two scientific consensus documents that concluded that ETS exposure is harmful to health. In short, this study gauges the number, sponsorship, authorship, and quality of symposia on ETS."

"We identified 11 symposia on the topic of ETS; four of these explicitly acknowledged tobacco industry funding. Although seven symposia did not acknowledge industry sponsorship, at least two of these were organized by individuals who are affiliated with the tobacco industry."

"Fifty-nine percent (175/297) of symposium articles presented original research and 41% (122/297) were review articles, compared with 90% (90/100) of journal articles that presented original research."

"[A] larger proportion of symposium articles than journal articles agreed with the tobacco industry's position."

"The distribution of topics of articles on ETS differed significantly between the symposium articles and the journal articles. The journals contained a larger proportion of articles that assessed the health effects of ETS, including epidemiologic studies and animal studies. The symposia contained a larger proportion of articles that assessed the effects of factors that could confound an association between ETS and tobacco-related diseases (eg, cooking smoke, radon, and diet)."

"[A] higher proportion of symposium authors than journal article authors were affiliated with the tobacco industry."

"[A] larger proportion of articles from symposia that acknowledge industry funding agreed with the tobacco industry position that ETS is not harmful than articles from symposia that did not acknowledge funding. Articles from symposia that did not acknowledge funding were more likely to focus on confounding factors than articles from industry-sponsored symposia."

"[T]he symposium authors published a lower proportion of peer-reviewed journal articles than the authors of the consensus documents....While 85% (23/27) of the consensus document authors were affiliated with academic institutions, 36% (5/14) of the authors who frequently wrote symposium articles were from universities. In contrast, 43% (6/14) of the symposium authors were consultants to the tobacco industry and one worked for the tobacco industry, while none of the authors of the consensus reports were in similar positions."

"The content of symposia on ETS differs from the content of journal articles on ETS in ways that suggest that symposia are not balanced and that they present the tobacco industry position on ETS. Symposia consist, in large part, of review articles that reach conclusions that are contrary to independent scientific consensus documents and meta-analyses published in journals. Symposium articles minimized the potential effects of ETS on health by claiming that ETS exposure is impossible to measure, or if it can be measured, levels of exposure are not high enough to produce physiological effects....Symposium articles contained a greater emphasis on potential confounding factors than journal articles. Most of the symposium articles, however, exclude ETS from comparative analysis of other health risk factors such as exposure to radon, cooking fuel, or automobile emission. For example, most articles discussing sick building syndrome did not mention ETS as a component of indoor air."

"The scientific reliability of the journal articles or consensus documents should be considered superior to that of symposia."

"Our data suggest that industry control over publication, rather than industry funding for research, is likely to influence the presentation of findings."

"The finding that data and reviews suggesting that ETS is not a health hazard are published in non-peer-reviewed literature has substantial implications for public policies regarding smoking restrictions. In June 1993, the US Supreme Court ruled that federal judges must ensure that scientific evidence and testimony admitted in trials are reliable. Whether the data have been subjected to peer review and publication is one criterion that judges are expected to consider. This ruling is directly relevant to the lawsuit that the tobacco industry has recently filed against the Environmental Protection Agency (EPA) claiming that the EPA used inaccurate and incomplete data for its risk assessment of ETS. As the court hears this case, it will have to decide whether non-peer-reviewed, tobacco industry-sponsored research, such as that presented in symposium proceedings, is admissible as evidence."

"Sponsored symposium proceedings influence public policy because they are often presented in a misleading fashion, as if they are equivalent to peer-reviewed journal articles, as if they are balanced reviews of the scientific literature, and as if they are not affiliated with the tobacco industry."

"The first tobacco industry-sponsored symposium occurred in 1974, well before journal articles on ETS were published. This suggests that the tobacco industry was concerned about adverse health effects of ETS before the medical community had conducted much research on the topic."

"Data from symposia should be reviewed skeptically since their funding sources may not be acknowledged, they have not been peer-reviewed, and they may not present a balanced overview of the scientific literature.

APPENDIX B

UPCOMING SCIENTIFIC MEETINGS

• **March 4, 1994**
Indoor Air Quality: An Overview for People Who Need to Know, AIHHM, San Antonio, Texas [Issue 57, Item 35] Same program to be held April 13, 1994, Minneapolis, Minnesota; May 5, 1994, Chicago, Illinois; June 17, 1994, Oklahoma City, Oklahoma; July 14, 1994, Anchorage, Alaska

• **March 8-10, 1994**

Orientation to Indoor Air Quality, U.S. Environmental Protection Agency, Birmingham, Alabama [Issue 65, Item 25]

• **March 12-16, 1994**
Conference, Environmental Information Association, Mission Valley, California [Issue 65, Item 26]

• **March 14-16, 1994**
Indoor Air Pollution, University of Tulsa, Tulsa, Oklahoma [Issue 65, Item 27]

• **March 22-24, 1994**
Indoor Environment '94, IAQ Publications and other sponsors, Washington, D.C. [Issue 61, Item 30]

• **March 28-31, 1994**
Eleventh ORNL Life Sciences Symposium, Indoor Air and Human Health Revisited (Bringing Selected Advances in Medical Science to the Indoor Air Quality Community), Knoxville, Tennessee [Issue 58, Item 43]

• **May 5-7, 1994**

Second Annual IAQ Conference and Exposition, NCIAQ, Tampa, Florida [Issue 49, Item 35]

• **May 22, 1994**
Indoor Air Quality Symposium, American Industrial Hygiene Conference and Exposition, Anaheim, California [Issue 57, Item 34]

• **August 22-25, 1994**
Healthy Buildings '94, Budapest, Hungary [Issue 63, Item 26]

• **September 7, 1994**
One-Day IAQ Course, Environmental Law and Policy Program, George Washington University, Washington, D.C. [Issue 63, Item 25]

• **October 10-14, 1994**
9th World Conference on Tobacco and Health, Paris, France [Issue 60, Item 38]

• **October 18-20, 1994**
Indoor Air Quality in Asia, Beijing, China [Issue 54, Item 42]

• **October 30-November 2, 1994**
IAQ '94: Engineering Indoor Environments, ASHRAE and other sponsors, St. Louis, Missouri [Issue 58, Item 42]

ETS/IAQ Report
Fax Communication Sheet

NOTE: If you would like additional information on one of the stories contained in this issue, or if you have information or ideas that we could incorporate into future issues, please complete and FAX this form.

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